## Editorial An Unhealthy Start in Life— What Matters Most?

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Life isn't fair. The following monograph describes how neurotoxic metals, micronutrient deficiencies, and social environments can combine to give a child an unhealthy start in life. Visualize a preschool child from a low-income family in the United States, growing up in a poorly maintained older apartment with peeling paint and windowsills laden with lead dust. Perhaps this child also attends a day-care center with a high child-to-caretaker ratio that prevents the caretakers from spending much time interacting with the children, and where the children often squabble over the toys that are present. Because of the high child-to-caretaker ratio, the caretakers are relatively harsh in their discipline. The child's parents both work, but at low-wage part-time jobs without health insurance or sick leave. The family's food stamps run out each month, and only one visit to the food pantry per month is allowed. The child has no picture books and few age-appropriate toys because the family has changed residence three times in the past 2 years. Unable to pay the rent, they lived in a homeless shelter once while between residences, with no place to store their personal belongings.

The scenario I have described above is real life for too many American children. We know that a child growing up with poor nutrition, lead or other toxic exposures, low-quality day care, and little stimulation in the home is getting an unfair start in life. But which of the factors in this child's life are the most influential? Perhaps that guestion is too simple: The problem is that risk factors often occur in an intercorrelated complex that cannot be untangled easily by statistical control. This monograph, by the interdisciplinary team of Hubbs-Tait, Nations, Krebs, and Bellinger, invites us to think more deeply about how the effects of metallic neurotoxicants, deficiency in micronutrients, and aspects of the social environment can, separately and together, influence children's behavioral health. The effects of combinations of risk factors on children's development have long been a puzzle to developmental researchers. One solution is to say, "Risk factors add up, so more risks are worse-regardless of the type of risk." Certainly there is truth in that answer, but this monograph sets the stage for new inquiries, with the potential to identify both protective factors as well as combinations of events that potentiate each other's negative effects. For example, does iron deficiency make lead exposure more deleterious to a child's cognitive functioning? Alternatively, does treatment of iron or zinc deficiency imply that lead will have weaker effects on a child's cognition or behavior?

The authors make the case that the effects of all three categories of influence may be subject to "effect modifiers," or interactions that exacerbate (or ameliorate) their influence. If we are to identify and establish preventative programs for children who are most susceptible to toxic exposures, micronutrient deficiency, and poor social environments, then we need to consider interactions. The authors also point out that unless all three sources of influence are considered, we could incorrectly attribute stronger or weaker influence to any one individual factor. For example, in epidemiological studies of the effects of neurotoxicants such as lead and mercury, investigators partial out the effects of social variables such as income, social quality of the home, parental education, and racial or ethnic identity of the parents or child. But it is rather unusual for studies of the impact of social influences such as parental sensitivity, parental education, or socioeconomic status to statistically control for either neurotoxic exposures or nutritional status in the child. Because of these standard research practices, estimates of the effects of social factors are likely overestimated, as are the effects of micronutrients.

The report is also cutting edge in the way it combines animal research on the mechanisms by which toxic metals, micronutrients, and environmental stimulation affect neural function and development with epidemiological research on children's cognitive and behavioral outcomes. Some of the latest work on neurotransmitter systems and their complex interactions is reflected here. For example, manganese is both a micronutrient and, at higher exposures, a neurotoxicant. Manganese affects glutamate uptake in certain areas of the brain, but it also affects dopaminergic systems. Lead can also alter glutamate neurotransmitter systems that are known to be involved in learning. These kinds of findings help us understand why lead and manganese are neurotoxic and what behavioral effects to look for. But such findings also raise the issues of coexposure to toxic substances and how micronutrients combine with neurotoxicants. The report calls for research that will address exposure to manganese and cadmium because these neurotoxicants are understudied while environmental releases of them are increasing. The current situation with respect to cadmium and manganese is reminiscent of how lead and mercury were regarded 40 or so years ago. Before the methylmercury poisoning disaster at Minamata, Japan, and before follow-up studies of children who were lead-poisoned were conducted, there was little societal concern about environmental releases of either lead or mercury. If we don't attack the research tasks set out by Hubbs-Tait and her colleagues in this monograph, we won't know until it is too late.